EP_A007

WHEN NSTEMI IS NOT CORONARY DISEASE: MINOCA REVEALING PHEOCHROMOCYTOMA

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INTRODUCTION

Pheochromocytoma is a catecholamine-secreting tumor with diverse cardiovascular manifestations, including myocardial infarction with non-obstructive coronary arteries (MINOCA). We report a case of biochemically confirmed pheochromocytoma initially presenting as non-ST-elevation myocardial infarction (NSTEMI), later reclassified as MINOCA.

CASE

A 62-year-old female with type 2 diabetes mellitus and hypertension was admitted with presumed NSTEMI and commenced on dual antiplatelet therapy. Further history revealed recurrent presyncope associated with paroxysmal headache, palpitations, and profuse diaphoresis. During admission, her blood pressure was markedly labile, ranging from 75/45 to 220/122 mmHg, raising suspicion of pheochromocytoma. Biochemical evaluation demonstrated markedly elevated 24-hour urinary normetanephrine of 36.19 µmol/day (reference 0–2.13) and methoxytyramine of 3.90 µmol/day (reference 0.10–1.79), consistent with catecholamine excess. Dedicated adrenal computed tomography identified a 3.8-cm right adrenal lesion with high attenuation (44 Hounsfield Unit [HU]), arterial enhancement (119 HU), and low washout (absolute 40%, relative 24%), without calcification or necrosis. Electrocardiography showed sinus rhythm with T-wave inversion in leads I, aVL, and V5–V6. Transthoracic echocardiography demonstrated a preserved left ventricular ejection fraction of 67% without regional wall-motion abnormalities. Coronary angiography subsequently showed normal coronary arteries, supporting a diagnosis of MINOCA likely secondary to pheochromocytoma-related catecholamine excess and hypertensive crisis. Antiplatelets were discontinued. She was commenced on α-blockade, with additional felodipine and low-dose β-blocker for blood pressure optimization, and subsequently underwent successful open right adrenalectomy. Postoperatively, she required transient inotropic support but was weaned within 36 hours.

CONCLUSION

Pheochromocytoma-associated MINOCA is uncommon but important to recognize. Catecholamine surges may cause myocardial injury through coronary vasospasm, myocardial oxygen supply-demand mismatch, and direct catecholamine-mediated cardiotoxicity. Recognition is crucial, as management differs fundamentally from atherosclerotic acute coronary syndrome and requires α-blockade before β-blockade.

EP_A008

THE DOPAMINE DILEMMA: EXOGENOUS L-DOPA OR RARE DOPAMINOMA?

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INTRODUCTION

Dopaminomas or dopamine-secreting pheochromocytomas are rare neuroendocrine tumors that frequently lack the classic paroxysmal symptoms of catecholamine excess (headache, sweating, palpitation). Diagnosing these tumors is exceptionally challenging when biochemical markers—specifically markedly elevated urinary dopamine—are interpreted in patients receiving exogenous Levodopa (L-DOPA) therapy, as the intake obscures clinical significance.

CASE

A 76-year-old male with ischemic heart disease, stage 4 chronic kidney disease, and new-onset hypertension was admitted for acute cholecystitis. Imaging studies incidentally revealed bilateral adrenal masses and demonstrated lipid-rich characteristics upon further evaluation with computed tomography (CT) adrenal washout. Biochemical evaluation revealed extreme elevations in 24-hour urinary dopamine (47,534 nmol/24 hours; normal <3,237) and 3-methoxytyramine (18.93 µmol/24 hours; normal <2.60), whereas downstream urinary noradrenaline (5.0 nmol/24 hours; normal 71.5–505.3), adrenaline (4.0 nmol/24 hours; normal 9.2–122.3), normetanephrine (0.22 µmol/24 hours; normal 0.88–2.88), and metanephrine (0.25 µmol/24 hours; normal 0.33–1.53) levels were all significantly below their respective reference ranges. The diagnosis was complicated by the patient's concurrent use of L-DOPA/Benserazide for flupentixol-induced parkinsonism. L-DOPA is a direct precursor to dopamine; its administration can cause massive, false-positive elevations in urinary dopamine, mimicking a dopaminoma's biochemical signature. However, the unique combination of profoundly high dopamine alongside suppressed downstream catecholamines and metanephrines suggested a true dopamine-secreting tumor—potentially secondary to dopamine beta-hydroxylase (DBH) deficiency—rather than drug interference.

CONCLUSION

This case illustrates the profound difficulty in diagnosing dopaminoma when exogenous L-DOPA therapy creates a near-identical biochemical profile. The diagnostic dilemma is further amplified by vague symptomatology, multiple comorbidities, and non-suggestive imaging. However, the finding of isolated dopamine hypersecretion with suppressed metanephrines serves as a critical clinical clue. This pattern points toward an intratumoral biosynthetic defect rather than drug interference. Clinicians must maintain a high index of suspicion and perform meticulous biochemical fractionation to identify these rare, dopamine-isolated secreting tumors.

EP_A009

THE HEMODYNAMIC PARADOX: SYNCHRONOUS ROBOTIC SURGERY FOR NORMOTENSIVE PHEOCHROMOCYTOMA IN VHL

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INTRODUCTION

Normotensive pheochromocytomas in Von Hippel-Lindau (VHL) syndrome present unique perioperative challenges. Standard alpha-blockade may induce intolerable orthostatic hypotension, making calcium channel blockers (CCB) a practical alternative. Furthermore, the primary intraoperative danger in these specific phenotypes may not be a hypertensive crisis, but profound vasoplegia. We report a VHL patient undergoing synchronous robotic surgery exhibiting this paradoxical hemodynamic response.

CASE

A 37-year-old female with VHL syndrome presented with an incidental 3.5-cm left adrenal mass and bilateral renal masses. Biochemistry confirmed a normotensive, noradrenergic pheochromocytoma (24-hour urine normetanephrines 4.2x upper limit of normal). Renal biopsy revealed a clear cell papillary renal cell tumor. Due to prior severe intolerance to Prazosin (hypotension/dizziness with low-dose Prazosin 0.5 mg ON), we utilized amlodipine for preoperative optimization. She was only able to tolerate low-dose 2.5 mg OD alongside oral sodium chloride and ample oral fluid loading. She underwent a synchronous robotic-assisted left adrenalectomy and left midpole renal tumor excision. Strikingly, tumor manipulation did not precipitate a hypertensive crisis. Instead, she developed hypotension requiring an intravenous noradrenaline infusion prior to adrenal vein ligation and tumor removal. Vasopressor support was successfully weaned 12 hours postoperatively, and she was discharged well.

CONCLUSION

Normotensive, noradrenergic pheochromocytomas in VHL are hemodynamically fragile. Chronic catecholamine excess induces homologous desensitization and downregulation of alpha-1 adrenergic receptors. This physiological adaptation explains the normotensive presentation and highlights the intraoperative vasoplegia experienced once sympathetic tone is altered by anesthesia. While CCB monotherapy with volume expansion safely facilitates prolonged, synchronous robotic surgeries, clinicians must anticipate and combat refractory hypotension rather than classical hypertensive spikes.

EP_A010

DISSEMINATED HISTOPLASMOSIS PRESENTING AS ADDISONIAN CRISIS: A DIAGNOSTIC MIMIC OF TUBERCULOSIS WITH BILATERAL ADRENAL MASSES

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INTRODUCTION

Disseminated histoplasmosis is a rare but important cause of adrenal insufficiency (AI), particularly in tuberculosis (TB)-endemic regions, where it may mimic granulomatous diseases. Adrenal involvement occurs in up to 80% of disseminated cases, although overt AI is less common. Reported cases described bilateral adrenal masses mimicking malignancy or TB, even in immunocompetent individuals. Addisonian crisis may be the initial manifestation, especially when the diagnosis is delayed. In TB-endemic settings, fungal infections are often overlooked, leading to delayed diagnosis and inappropriate therapy.

CASE

We reported a case of a 68-year-old male with underlying diabetes mellitus who presented with fever, cough, dysphagia, and weight loss for 1 month. He was empirically treated as smear-negative disseminated TB. On day 2 of therapy, he developed hypotension (80/50 mmHg), hypoglycemia (3.9 mmol/L), hyponatremia (Na 129 mmol/L), and hyperkalemia (K 5.1 mmol/L), suggestive of adrenal crisis, and was started on intravenous hydrocortisone. Serum cortisol prior to treatment was 61